

Case Report

Double coronary artery thrombosis presenting as acute extensive anterior ST-segment elevation myocardial infarction

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Abstract

Simultaneous thrombosis of more than one coronary artery is an uncommon angiographic finding in acute ST-segment elevation myocardial infarction (STEMI), and usually leads to cardiogenic shock or even sudden cardiac death. We reported a 56-year-old man presenting with persistent chest tightness and ST-segment elevation over precordial leads in electrocardiography (ECG). Emergent coronary angiogram showed total occlusion of both the proximal right coronary artery (RCA) and the proximal left anterior descending artery (LAD). We performed thrombus aspiration and stenting over the LAD with thrombolysis in myocardial infarction (TIMI) III flow to the distal LAD. However, diminishing collateral flow to the distal RCA complicated with complete atrioventricular block (CAVB) and cardiogenic shock developed thereafter. Because distal embolization of the collateral circulation from the LAD to the distal RCA was suspected, thrombus aspiration and stenting over the proximal RCA were performed. After reperfusion of the RCA, the patient's hemodynamic status stabilized and he recovered uneventfully.

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1. Introduction

Acute myocardial infarction (AMI) is typically caused by disruption of atheromatous plaques, resulting in thrombus formation leading to partial or complete occlusion of the coronary artery. Multivessel coronary disease is relatively common in patients with ST-segment elevation myocardial infarction (STEMI), with a reported prevalence ranging from 50% to 80% in selective populations.^{1,2} Although multiple ruptured plaques with thrombus formation have been reported in more than 10% of autopsied cases,³ concomitant thrombosis and complete occlusion of two coronary arteries is still an uncommon finding in angiography. Most patients with acute multivessel thromboses are critically ill and may present with

cardiogenic shock or sudden cardiac death before arriving to the hospital. We report a case of double coronary artery thrombosis that was successfully treated with thrombus aspiration and stenting.

2. Case report

A 56-year-old man presented to the emergency room with persistent severe retrosternal chest tightness and cold sweating for 1 day. Upon arrival, his vital signs showed a regular heart rate of 96 beats/minute, blood pressure of 117/89 mmHg, and mild respiratory distress with a respiratory rate of 24 breaths/minute. Physical examination showed decreased S1 intensity, Grade II/VI systolic murmurs over the left lower sternal border, and fine rales over the bilateral basal lung field on auscultation. The electrocardiography (ECG) on arrival showed sinus rhythm, a QS pattern, ST-segment elevation in lead V1–5, and Q wave in leads II, III, and aVF (lead augmented vector foot) (Fig. 1A). Chest radiography revealed

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Fig. 1. (A) Twelve-lead electrocardiography (ECG) on arrival to the emergency room showed a QS pattern and ST-segment elevation in V1–4 and Q wave in leads II, III, and aVF (lead augmented vector foot). (B) ECG tracing after successful coronary stenting of the left anterior descending coronary artery (LAD) showed complete atrioventricular block (CAVB).

cardiomegaly and mild cephalization of pulmonary vasculature. Laboratory studies revealed a white cell count of $12.4 \times 10^9/L$, hemoglobin concentration of 137 g/L, platelet count of $221 \times 10^9/L$, creatine kinase (CK) of 3272 U/L, CK-MB of 481 U/L, and troponin-I 107.4 $\mu g/L$. Other study results were unremarkable.

The patient's medical history revealed that he smoked one pack of cigarettes per day and was a social drinker. There was no past history of other systemic illnesses or family history of premature coronary artery disease. He mentioned an episode 2 years earlier of severe chest tightness, which subsided spontaneously without any medical management.

Prior to emergent coronary angiography, the patient was preloaded with dual antiplatelet therapy. It showed total occlusion with suspected thrombus formation of both the left anterior descending coronary artery (LAD) and the proximal right coronary artery (RCA), with collateral flow from the distal part of the left circumflex coronary artery (LCX) and LAD to the distal RCA and posterolateral (PL) branch (Fig. 2A–C). Left ventriculography revealed an ejection fraction of 28% with a sizeable apical aneurysm, and

dyskinesia over the anterior and inferior walls of the left ventricle. After engaging the left main coronary artery by use of a guiding catheter, transient ventricular fibrillation developed and subsided after five defibrillations. A Fetch Aspiration Catheter (Medrad Inc., Warrendale, PA, USA) was used for thrombus aspiration and several small pieces of red thrombus were aspirated. Then, an intracoronary loading dose of Tirofiban (Irokocardio international SARL, Geneva) was administered according to the patient's body weight. The thrombolysis in myocardial infarction (TIMI) flow of the LAD improved from 0 to II. After balloon dilatation, a Cypher (Cordis Corporation, Miami Lakes, FL, USA) 2.5 mm \times 33 mm drug-eluting stent was deployed over the middle to distal part of the LAD, and another Cypher 3.0 mm \times 28 mm stent over the proximal to middle part of the LAD. After the stenting procedure was completed, coronary flow over the LAD improved to TIMI III with good coronary brushing (Fig. 2D,E). However, bradycardia with complete atrioventricular block (CAVB) and hypotension developed progressively and a temporary pacemaker was placed. In addition, collateral flow from the LCX and the

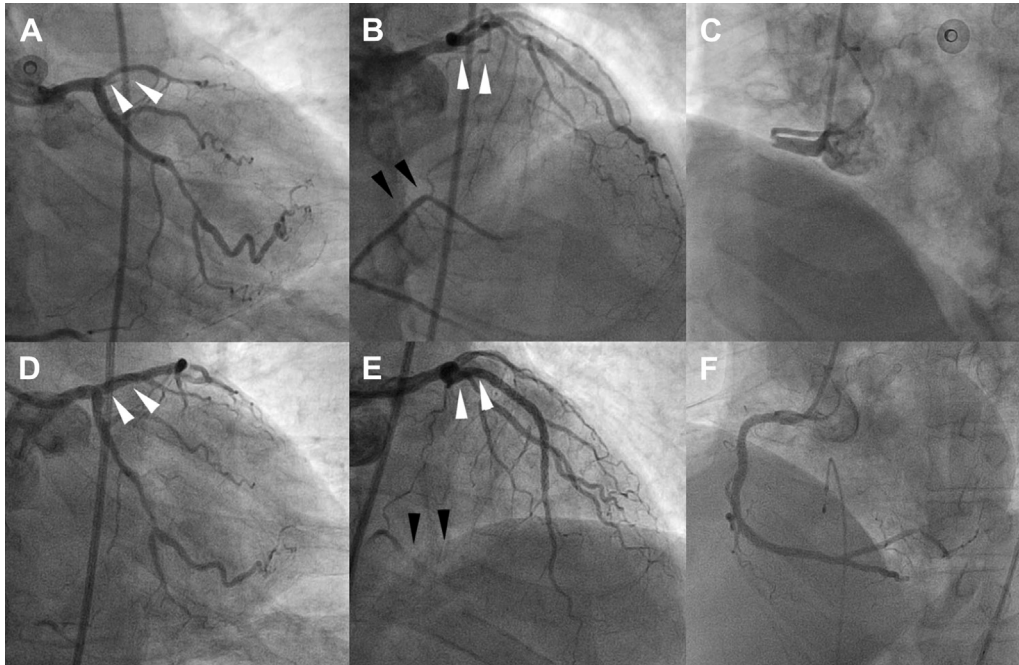


Fig. 2. (A) Total occlusion of the left anterior descending coronary artery (LAD) ostium with suspected large thrombus burden (white arrow). (B) Total occlusion of the LAD ostium (white arrow) and abundant collateral from septal branches and the left circumflex coronary artery to the right coronary artery (RCA)-posteriolateral and -posterior descending artery branches (black arrow). (C) Total occlusion of the RCA proximal part. (D) The final angiography result after thrombus aspiration and stenting of the LAD (white arrows indicate the LAD). (E) Final LAD angiography with diminished flow and contrast stasis over the RCA-posteriolateral and -posterior descending artery branches. (F) The RCA final angiography after thrombus aspiration and stenting.

LAD to the distal RCA, posterior descending artery, and PL branches was decreased with some contrast stasis (Fig. 2E). As thrombotic occlusion of the proximal part of the RCA was also suspected, we performed thrombus aspiration by Fetch aspiration catheter after prompt crossing of the RCA proximal occlusion by a floppy guidewire. The TIMI flow of RCA improved to II after thrombus aspiration. A Jostent (Abbott Vascular, Santa Clara, CA, USA) 4.0 mm × 26 mm bare metal stent was then deployed over the proximal to middle RCA with a final RCA TIMI III flow (Fig. 2F). After the procedure, the CAVB resolved and the heart rhythm returned to sinus. After the emergent procedure, the patient recovered gradually and was discharged under relatively stable conditions 9 days later, in spite of mild heart failure according to New York Heart Association Functional Class I–II.

3. Discussion

Rupture of unstable coronary plaques with subsequent thrombotic occlusion is a common pathologic finding in AMI. Multivessel coronary artery stenosis is commonly seen in primary coronary angioplasty in STEMI and it has an adverse impact on patient prognosis. However, a culprit-only revascularization strategy is suggested in these patients unless an unstable hemodynamic status is involved, or if signs of residual myocardial ischemia support nonculprit vessel intervention.⁴ By contrast, multivessel coronary artery thrombosis in STEMI is uncommon and always carries a catastrophic outcome. According to a review of 23 cases by Kanei et al, the

risk factors of multivessel coronary artery thrombosis included essential thrombocytosis, intravenous cocaine abuse, diabetes, and smoking. The LAD (78%) and the RCA (87%) were the arteries involved in most patients.⁵ The initial ECG is indispensable in localizing the infarct-related coronary artery, and most of the cases have clear manifestations of infarct-related ST-segment or T wave change in acute STEMI.⁶ However, some presented as ventricular arrhythmia.⁷

The pathophysiology of multivessel coronary artery thrombosis remains unclear. Goldstein et al showed the existence of additional unstable lesions other than the culprit lesions in 21% of patients with MI.⁸ Using an angioscope, Asakura et al found that yellow plaques were equally prevalent in the infarct-related and non-infarct-related coronary arteries in patients with acute MI, despite the fact that thrombus is more common in culprit lesions. Therefore, plaque instability with pancoronaritis in the AMI scenario causes a predisposition for development of multivessel coronary artery thrombosis. Nevertheless, the underlying mechanisms warrant further study.⁹

In this case, the initial ECG showed ST elevation in the precordial leads and Q wave in the inferior lead, which may suggest that the LAD was the culprit vessel. However, the presence of fresh thrombus aspirated from the RCA as well as the LAD, as well as the development of CAVB and the presence of reperfusion arrhythmia after stenting of the RCA argued against the possibility that the proximal RCA lesion was a chronic nonculprit lesion. It is intriguing that even though total occlusion of RCA and LAD were noted in the

initial coronary angiogram, cardiogenic shock did not occur at first but developed only after reperfusion of LAD. Observing that the patient suffered from chest pain for more than 1 day and had a CK level elevated to 3272 U/L upon initial ER presentation, we speculated that there might be two lesions: one in the LAD and one in the RCA, respectively. At first, plaque rupture and thrombus formation occurred in the proximal RCA lesion, leading to total occlusion of the RCA. However, the rapid recruitment of collaterals from the LAD and the LCX to the RCA limited the extent of the infarction over the inferior wall, thus causing a relatively small Q wave over the inferior leads. Unfortunately, inflammation of the pancoronary vessels led to subsequent plaque rupture and thrombus formation over the LAD, causing new ST-segment elevation over precordial leads. After stenting of the LAD, it was likely that the no-reflow phenomenon occurred. This impaired not only the collateral from the LAD to the RCA, but also the collateral from the LCX to the RCA (compare stasis of the LCX collateral to the RCA-PL branch in Fig. 2E), resulting in cardiogenic shock and CAVB, which resolved after we recanalized the RCA. Even though a distal protection device is not routinely needed in primary percutaneous coronary intervention, it should be considered in huge thrombus burden intervention and when the target vessel has abundant collaterals to other vessels. Finally, as the atrioventricular (AV) node and His bundle are supplied by the AV nodal artery of the RCA and the first two septal perforators of the LAD,¹⁰ another possible mechanism is that after long stenting of LAD, the flow from septal branches to the AV node were severely compromised, and thus CAVB was ensured. However, this might be less likely because during the period when the LAD was totally occluded with balloon dilatation, no CAVB was noted.

Although the exact mechanism of simultaneous double coronary artery thrombosis in this case is hard to prove, our

case highlights the importance of early recognition and reperfusion of each culprit lesion in the scenario of acute MI with multivessel coronary artery thrombosis.

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